Clinical Guidance

Paediatric Critical Care:
Acid Base Interpretation

Summary

This guideline is for the use by clinical staff who are interpreting acid base balance in critical care. Rather than focusing on bicarbonate which is a derived value, interpretation is aided by accounting for the contribution made to acid-base by: chloride, albumin and unmeasured acids.

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This clinical guideline has been produced by the South Thames Retrieval Service (STRS) at Evelina London for nurses, doctors and ambulance staff to refer to in the emergency care of critically ill children.

This guideline represents the views of STRS and was produced after careful consideration of available evidence in conjunction with clinical expertise and experience. The guidance does not override the individual responsibility of healthcare professionals to make decisions appropriate to the circumstances of the individual patient.

Change History		
Date	Change details, since approval	Approved by
June 2022	Guideline reformatted. Language simplified to try and make accessible for entire team.	ELCGC

Why Acid-base Matters

- Acid-base balance is disturbed during critical illness
- Rapid or significant changes in acid-base balance have multi-organ implications, negatively impacting morbidity and mortality
- Normal cell metabolism depends upon maintenance of blood pH within a tight “normal” range – pH7.35-7.45
- Once pH falls, processes such as oxygen delivery to cells, electrolyte control and cardiac contractility can be negatively impacted
- Normal metabolism results in continuous production of two main acidifying forces - hydrogen ions (H+) and carbon dioxide (CO_2)
- The process which serve to balance pH against the production of H+ and CO2 is a complex synergy of action including chemical buffers in the blood, buffering within erythrocytes and respiratory/renal/brain function
- This guideline provides a framework for understanding blood gases by explaining different approaches of acid-base physiology

Henderson-Hasselbalch formula (circa 1917)

$$pH = 6.1 + (HCO_3/pCO2mmHg \times 0.003)$$

- Traditional teaching on acid-base relies on the Henderson-Hasslebalch formula.
- It states that HCO_3 (metabolic component) and pCO_2 (respiratory component) can vary independently
- It assumes that bicarbonate is the only significant buffer for acidosis

Limitations:

- In clinical practice the base excess is used to “quantify” the magnitude of metabolic component of an acid base derangement.
- Bicarbonate and base excess are not *directly* measured (they are calculated from pH and pCO_2 so they are always coupled and dependent on these. For this reason they can’t be used as direct measures of acid base.
- Both bicarbonate and base excess do not correlate well *in-vivo* with acid base disturbance because they assume all other blood components and electrolytes are normal (Hb, Na, Cl, Albumin) which is rare in sick patients as electrolyte problems are common

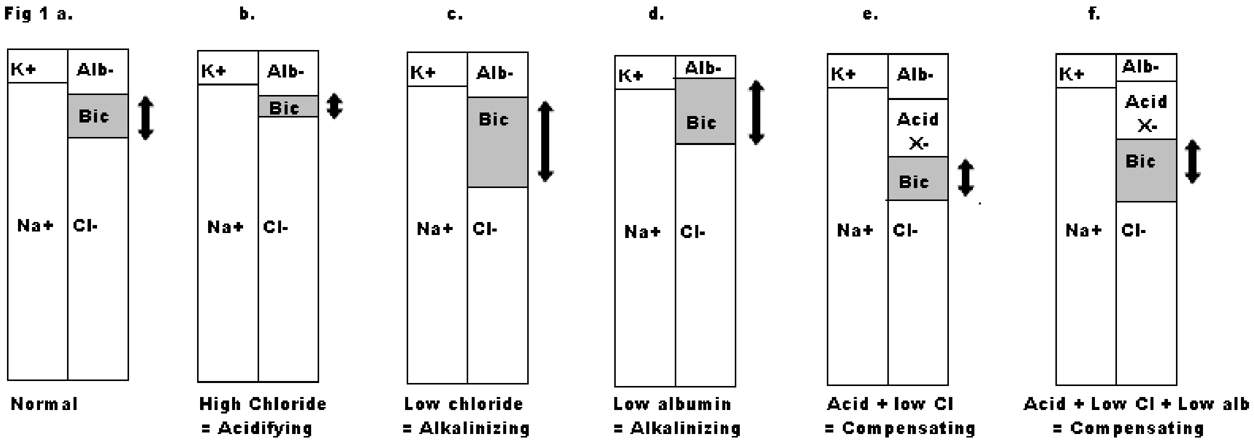
Stewart’s Strong Ion Methodology (circa 1981)

$$Na + K + Ca^{2+} + Mg^{2+} = Cl^{-} + HCO3^{-} + \text{lactate} + \text{albumin charge}$$

(Cation charge= Anion charge)

- Stewart defined the Strong Ion Difference (SID) as the absolute difference between completely dissociated anions and cations
- According to the principle of electrical neutrality, the SID is balanced by weak acids and CO_2
- Explains pH changes for all 3 major buffer systems
  1) Carbonic (pCO_2, bicarbonate)
  2) Electrolytes (Na, K, Cl^-, lactate, Ca, Mg)
  3) Weak acids (albumin, phosphate)
- Although complex, it can be simplified at the bedside to explain acid-base disturbances where electrolyte abnormalities are present, and is hence more accurate

1) If Cl^- is very high, bicarb is squeezed into a smaller space (hyperchloraemic acidosis) Figure 1b
2) If Cl^- is very low, bicarb has more space to occupy (hypochloraemic alkalosis) Figure 1c
3) If albumin is low, it will allow more space for bicarbonate (low albumin is alkalizing) Figure 1d
4) If albumin is high, it will reduce the space bicarbonate has (high albumin is acidifying)
5) If an anionic acid is present like lactate or ketones, a low Cl^- (Figure 1e) or in combination with a low albumin (Figure 1f) may partially buffer acidosis by allowing more space for bicarb to be in. This is common in clinical practice.



Normal

High Chloride = Acidifying

Low chloride = Alkalinizing

Low albumin = Alkalinizing

Acid + low Cl = Compensating

Acid + Low Cl + Low alb = Compensating

Stewart’s formula has been modified over the years to create a model familiar to many as the ***“anion gap”*** calculation

Role of Chloride in Acid-Base

1) Chloride: Sodium ratio

- Cl^- must always be interpreted relative to Na
- Normal Cl^- 106, Normal Na 140 → Cl:Na ratio = 0.75 or 75% (normal range 0.72-0.80)
    - Chloride is frankly acidifying if Cl >80% of Na
    - Chloride is frankly alkalinizing if cl <72% of Na

(NB Always consider Cl in relation to Na. Do not correct Cl for body water: it gives false values because Cl and Na do not distribute equally between ICF and ECF)

2) Partitioning base deficit for chloride and sodium (BE_{Cl})

- Effect of Cl and Na on the base excess can be simplified by this formula: **Base excess due to chloride and sodium = Na – Cl – 32**
- Example:
    - BE -10, Na 140, Cl 113
    - BE due to chloride = 140-113-32 = -5mEg/L
    - Chloride is acidifying by 5mEg/L or 50% of the base excess
    - The remaining 5mEg/L could be explained by other anionic acids – e.g. lactate, acids broken down from glycocallyx in sepsis

Role of Albumin in Acid-Base and The Anion-Gap

Anion Gap (AG) = Na – (Cl + HCO3)

- The Anion Gap (AG) is a derived variable used to determine the presence of unmeasured anions (normal range 4-12mmol/L) however it does not account for albumin being acidic.
- Albumin is a weak acid and has a charge in mEq/L of about 25% of the concentration in g/L (i.e. 40g/L has a charge of about 10mEq/L). The effect of albumin can be simplified by the following formula:

Albumin effect on base excess = (42 – Albumin g/L) x 0.25 = + **mEq/L**

- Therefore a low albumin is alkalinizing.
- **Rule of thumb:** Every 10 g/L fall of albumin will increase the base excess by 2.5 mEq/L
- Alternatively the effect of "unmeasured anions" can be quantified by the partitioned base excess by the difference between all known components of base excess (albumin and chloride) from the total base excess:

$$\textbf{Base Excess}_{\textbf{UNMEASURED}} = \textbf{SBE} - \textbf{BE}_{\textbf{CHLORIDE}} - \textbf{BE}_{\textbf{ALBUMIN}}$$

Acid-Base Made Simple – Clinical Application

- Define nature of disturbance (low pH = acidaemia, high pH = alkalaemia)
- Define what forces are acidifying and which are alkalinizing and how they balance

Component	Acidifying force	Alkalinizing force
Carbon Dioxide	↑ pCO_2	↓ pCO_2
Chloride	↑Chloride	↓Chloride
Albumin	↑Albumin	↓Albumin

Formulas

- Cl:Na >80% = frank hyperchloraemia
- Cl: Na <72% = frank hypochloraemia
- Base excess Cl = Na – Cl – 32
- Base excess albumin = (42 – Albumin g/L) x 0.25
- Base excess unmeasured anions

$$= BE_{total} - BE_{Cl} - BE_{Alb}$$

References:
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4) Sood et al, Indian J Crit Cre Med 20120 Apr-Jun; 14(2): 57-64
5) Life In The Fastlane – Acid base, Anion Gap, ABG interpretation